SPONTANEOUS COMPRESSION OF THE MEDIAN NERVE IN THE CARPAL TUNNEL

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Numbness, tingling and pain in the hands, sometimes associated with weakness and wasting of the small hand-muscles, is a common condition. Many cases of this symptom complex have been diagnosed, depending on the fashion at the time, as cervical rib, scalenus anticus, thoracic inlet or costo-clavicular syndromes. At the present time the popular diagnosis for similar cases is cervical disc disease. A common cause of this symptom complex is, however, compression of the median nerve under the flexor retinaculum in the carpal tunnel.

ANATOMY

The walls and floor of the carpal tunnel are formed by the carpal bones. The roof is formed by the flexor retinaculum which stretches from the pisiform bone and the hook of the hamate bone on the ulnar side to the tubercle of the scaphoid and the crest of the trapezium on the radial side.

Through the carpal tunnel pass the tendons of the long flexors of the fingers and thumb, the tendon of flexor carpi radialis and the median nerve, which lies immediately below the flexor retinaculum, superficial to the flexor tendons.

Across the flexor retinaculum pass the ulnar vessels and nerve and the palmar cutaneous branches of the median and ulnar nerves. At the distal border of the flexor retinaculum the median nerve gives off, on its radial side, a stout recurrect motor branch which supplies the abductor pollicis brevis and the opponens pollicis and in 20% of cases the flexor pollicis brevis. The median nerve then splits into branches to supply the first and second lumbricales and the skin on the palmar aspect of the radial 3½ digits as well as the skin on the dorsal aspect of these digits distal to the proximal inter-phalangeal joint.

The palmar cutaneous branch of the median nerve (which passes over the flexor retinaculum) supplies the skin over the thenar eminence and the skin of the palm on the radial side of a line drawn along the middle of the long axis of the metacarpal of the ring finger.

CAUSES OF COMPRESSION

Changes in the bones forming the floor and walls of the carpal tunnel may cause compression of the median nerve by decreasing the size of the carpal tunnel. Compression has thus been described as a complication of fracture of the lower end of the radius,[2-5] fracture of the carpal bones,[2,5] dislocation of the lunate[6] and osteoarthritis of the wrist.[5]

An increase in the amount of soft tissue in the carpal tunnel may compress the median nerve. Compression has been due to a cyst arising from the flexor tendon sheaths,[5] to deposits of amyloid tissue in the carpal tunnel in both primary and secondary amyloidosis,[7] and to spread of infection from wounds in the hand to involve the flexor tendons in the carpal tunnel.[8] An abnormal artery or an abnormal slip of the flexor digitorum sublimis has been known to compress the median nerve.[9]

Cases of acromegaly [10] have shown the typical picture of this syndrome. Here the condition may be due to an increase in the amount of soft tissue in the carpal tunnel or to arthritic changes.

In 3 reported cases compression of the median nerve was associated with rheumatoid arthritis,[7,11] but in these cases it was not clear whether the rheumatoid arthritis was responsible for the compression, or whether the compression was a coincidental occurrence.

Cannon and Love[4] were the first to recognize that compression of the median nerve in the carpal tunnel can occur without any obvious local pathological change or any generalized disease to account for the compression. Such cases are referred to as cases of spontaneous compression and, although only recently recognized for the first time, they by far outnumber the cases in which an underlying pathological change can be found.

MECHANISM OF SPONTANEOUS COMPRESSION

Active sharp flexion of the wrist and fingers pulls the flexor tendons tightly against the flexor retinaculum so that the median nerve may become compressed during this movement. Phalen[12] believes that sharp flexion of the wrist narrows the space between the radius and the flexor retinaculum, so that the median nerve gets compressed between the proximal edge of the flexor retinaculum, and the radius. Meadoff's work[13] is in support of the flexion theory. He injected lipiodol into the sheath of the median nerve and demonstrated that it flows freely into the palm with the wrist in extension, but stops sharply at the level of the flexor retinaculum with the wrist in flexion.

Against the flexion theory must be stated the work of Brain *et al.*,[14] who noted that there is more room in the carpal tunnel with the wrist in flexion than in extension, and by direct measurement they found the pressure in the carpal tunnel to be much higher with the wrist in extension than in flexion. They believe that an occupation which calls for repeated extension of the wrist is an important factor in causing compression of the median nerve. It must further be borne in mind that repeated sharp flexion of the wrist is an uncommon

movement in most occupations, whereas repeated extension is a very common movement.

The relative importance of flexion and extension is difficult to access because the conditions prevailing in the carpal tunnel during active movement of the wrist must be different to those prevailing during passive movement in the anaesthized patient and in the cadaver.

Mayer and Barry[15] believe that repeated small injuries at the wrist are the etiological basis for the development of compression but they are uncertain of the mechanism.

Michaelis[11] believes that occupational overstrain on the palmaris longus can cause thickening and rigidity of the flexor retinaculum where the palmaris longus is inserted into it. This change may progress to such an extent that compression of the median nerve may result. He points out that this change has not been noted by previous workers because the distal part of the flexor retinaculum, where the palmaris longus is inserted, cannot be seen with the usually employed transverse skin incision at the wrist. To observe the changes as described by him the flexor retinaculum must be fully exposed through a skin incision extending into the palm.

The work of Denny-Brown and Brenner[16] shows that pressure on a segment of nerve, sufficient to render the segment ischaemic, will cause the nerve above and below the area of pressure to become oedematous. Should compression of the median nerve occur in the carpal tunnel the resulting swelling of the nerve will further increase pressure and so a vicious circle will result.

Kremer *et al.*[5] believe that transient ischaemic compression of the median nerve may result from swelling of the soft tissues in the carpal tunnel. Such swelling will tend to occur at night owing to vascular stasis from lying on the arm and from CO_2 retention during sleep with consequent vasodilatation and increased production of tissue fluid.

It is tempting to invoke an endocrine disturbance but if the endocrines play any role, the role is obscure. Most cases occur in menopausal women but men are not immune. Some cases developing during pregnancy have a spontaneous remission following delivery[17] and may be caused by fluid retention. Cases occuring in association with acromegaly are probably due to pathological changes at the wrist and not to a direct hormone action.

SIGNS AND SYMPTOMS OF SPONTANEOUS COMPRESSION

The condition is commonest between the ages of 40 and 50 years but it may occur as early as 26 or as late as 68 years.[5]

Females are affected at least 5 times more frequently than males.[5] They are often busy housewives who do their own housework.

As stated above, the condition may arise during pregnancy and relief may follow delivery.[17]

In the great majority of cases the condition starts in the dominant hand. There is a strong tendency for the other hand to become involved subsequently, so that sooner or later most cases are bilateral. [5, 12, 14, 17]

The history usually extends over a few years but may be as short as 2 weeks[17] or as long as 28 years.[5]

The onset is usually insidious, with paroxysmal nocturnal attacks of paraesthesia in the fingers or hand. A burning, tingling, numb sensation may waken the patient at night or be present on awakening in the morning. As the attacks of paraesthesia become more severe, pain accompanies them. At this stage attacks of paraesthesia and pain may also come on during the day but are never so frequent or so severe as at night.

The patients usually find it difficult to locate the paraesthesia and pain accurately and often refer the symptoms to all the digits or the whole hand. Kremer *et al.*[5] found that at the first examination only about half of their patients localized the symptoms to the median nerve distribution in the hand. When the patients were instructed to note the distribution of the symptoms during attacks very carefully, they found that few of the patients admitted to symptoms in the little finger.

When attacks of paraesthesia are frequent and severe, most of the patients will complain of accompanying agonizing, deep-seated, burning, aching, bursting pain. The pain is at its worst in the fingers and deep in the palm but is often felt deep in the muscles of the inner aspect of the forearm as far as the elbow and some cases have pain even as high as the shoulder.[5]

The patient may get relief by hanging the hand out of the bed, by rubbing or shaking it or by holding the wrist straight. The discomfort may, however, be so severe that the patient has to get up and walk about. Often the patients are afraid to go to bed at night so that, in addition to pain, the patient may now begin to suffer from lack of sleep.

During the attacks of paraesthesia and pain the fingers and hand feel swollen, useless and stiff, but objective swelling is very rarely noted.[5]

At this stage or before it is reached the patient may notice that there is a blunting of sensation on the finger tips and that the hand becomes clumsy and weak, especially in holding small objects. Pain may now begin to diminish and sensory and motor loss become more conspicuous, but some patients have severe attacks of paraesthesia and pain for many years without developing abnormal motor or sensory signs.

Objective motor and sensory signs are limited to the hand and are never found proximal to the wrist. They are late in development and are an indication of advanced damage to the median nerve.

Blunting of sensation is inconspicuous and is limited to the median-nerve distribution in the fingers. The change is most marked in the finger-tips but may also be found on the palmar aspect of the fingers. It is rare for all $3\frac{1}{2}$ median-nerve-supplied digits to be affected. The tip of the middle finger is almost always involved to the greatest degree. Because the palmar cutaneous branch of the median nerve lies superficial to the flexor retinaculum it is not compressed and hence there is no sensory loss over the thenar eminence and palm.

When there is clumsiness and weakness of the hand, wasting may be found of the abductor pollicis brevis alone or with the opponens pollicis. The flexor pollicis brevis is usually spared, for it is supplied by the ulnar

nerve in 80% of cases. Fibrillary tremor is not observed in the wasted muscles.[4]

Colour changes in the hands were mentioned by 5 of the 40 cases reported by Kremer *et al.*[5] These changes were not necessarily related to the attacks and were never observed by the authors themselves.

The transient ischaemia test described by Gilliat and Wilson[18] is a very helpful diagnostic aid. It depends on the arrest of circulation to the arm by means of a pneumatic tourniquet above the elbow. In patients with compression of the median nerve in the carpal tunnel one may find:

(*a*) Within 30-60 seconds of arrest of circulation an intense tingle and sharp pricks would start in the thumb, index and middle fingers and perhaps in the ring finger; it may spread to the thenar pad and palm or above the wrist to the radial side of the arm; it never spreads to the little finger; patients may develop a 'tight' or 'bursting' pain similar to their spontaneous pain at night. This characteristic altered intense paraesthesia and pain has only been found with median-nerve lesions at the wrist and only when severe irritation has been present.

(*b*) Within 5-10 minutes of arrest of circulation median sensory loss may appear, so that easily detectable hypaesthesia of all digits supplied by the median nerve is demonstrable. This early ischaemic sensory loss in the median-nerve territory may occur with a lesion of the median nerve at any level above or below the tourniquet round the arm. It indicates a lesion of the median nerve but does not localize it as the occurrence of altered paraesthesia does.

Tinel's test, which is a tingling sensation radiating out into the hand obtained by light percussion over the median nerve at the wrist, is of doubtful value in diagnosis. Phalen[12] found this test positive in every one of his 11 cases, whereas Kremer *et al.*[5] found it positive in only 2 of their 40 cases.

Occasionally a tender swelling is visible or palpable just above the flexor retinaculum.[12, 15, 17] At operation this is found to be the oedematous median nerve. Such a swollen median nerve is often referred to as a pseudo-neuroma. A true neuroma is very rare.

In a few patients it is possible to produce or increase the paraesthesia and pain in the hand by acute flexion or extension of the wrist and maintenance of the wrist in that position for 60 seconds.[12, 15]

The clinical picture of compression of the median nerve secondary to obvious pathological changes at the wrist does not differ significantly from that of spontaneous cases, except that symptoms are confined to the pathological side and are thus usually unilateral, and that diurnal attacks are more likely to be brought on by the use of the hand.[17]

DIFFERENTIAL DIAGNOSIS

The thoracic inlet, costo-clavicular, cervical rib and scalenus anticus syndromes, cervical disc disease and cervical osteo-arthritis are unlikely to cause paroxysmal nocturnal attacks of paraesthesia and pain. Thenar atrophy, which may occur in these conditions, is unlikely to be associated with cutaneous sensory loss limited to the median-nerve distribution in the digits. Pain extending from the hand into the arm even as high as the shoulder may be due to compression of the median nerve in the carpal tunnel, so that this distribution of pain does not necessarily indicate a lesion higher than the wrist.

In polyneuritis the paraesthesia will not be paroxysmal and there will not be nocturnal attacks of pain. If sensory changes are present they will not be confined to the median-nerve distribution in the digits.

Lesions of the median nerve proximal to the wrist can usually be recognized because of involvement of muscles of the forearm.

In ulnar-nerve lesions the symptoms are unlikely to occur in nocturnal attacks and the signs are localized to the ulnar-nerve distribution.

Progressive muscular atrophy, amyotrophic lateral sclerosis and syringomyelia can be easily differentiated on the basis of the more widespread neurological involvement occuring in these conditions.

Painful arthritis of the wrist is not a differential diagnostic problem but it may give rise to secondary compression of the median nerve.

Once compression of the median nerve in the carpal tunnel has been diagnosed it is not difficult to separate the spontaneous cases from those due to bony changes at the wrist, to infection in the flexor tendon sheaths, or from those which occur in association with sytemic diseases such as acromegaly, amyloidosis or rheumatoid arthritis.

TREATMENT

Very mild cases of short duration may be treated by resting the arm. Some patients notice that symptoms improve or even disappear while on holiday, only to relapse with resumption of normal work. Immobilization of the wrist by splinting may cause symptoms to disappear for long periods or in rare cases even permanently.

When a patient has had recurrent attacks, when symptoms are severe, or when there are signs of permanent injury to the median nerve as evidenced by muscle atrophy or sensory loss, decompression of the median nerve must be done by section of the flexor retinaculum. By far the majority of patients fall into this group. Phalen[12] goes so far as to urge operation in all cases.

The operation is best done under general anaesthesia in a bloodless field through a 1-inch-long transverse incision along the middle third of the distal wrist crease. Section of the flexor retinaculum is most conveniently done with the retinaculotome designed by Paine.[19] The flexor retinaculum must be sectioned along its medial border to minimize the possibility of damage to the recurrent motor branch of the median nerve. Care must also be taken not to damage the palmar cutaneous branch of the median nerve, which lies superficial to the flexor retinaculum. It is important to ensure complete division of the whole of the flexor retinaculum, particularly its distal fibres in the palm. The median nerve must not be dissected out at the wrist, for if this is done it is liable to become adherent to the vertical incision in the flexor retinaculum.

In the open operation the whole flexor retinaculum is exposed through a skin incision extending into the palm. This incision is liable to leave a bad scar, which may become adherent to the median nerve, and it should only be employed when it is essential to examine the entire flexor retinaculum in an open field.

After operation a pressure bandage is applied and the arm is elevated for 12 hours. The patient is sent home the day after the operation and is encouraged to use the fingers in light work such as knitting, sewing and writing.

In bilateral cases the most severely involved hand may be operated on first, or both hands may be operated on at the same time.

PROGNOSIS

Without operation relapses are the rule and permanent damage to the median nerve is liable to occur.[15]

With operation in early cases the condition is dramatically and permanently cured.[5, 12, 14, 15, 17] In late cases relief of pain and paraesthesia is still dramatic but hypaesthesia and atrophy may take up to 6 months or longer to clear up completely.[12] In very long-standing cases where the median nerve has been permanently damaged slight sensory loss and motor weakness and wasting may be permanent.

If after operation there is not satisfactory improvement the wrist must be explored again and the whole flexor retinaculum fully exposed. In a series of 40 operated cases Kremer *et al.*[5] had 3 failures. At the second operation it was found in one case that the distal fibres of the flexor retinaculum were undivided and still compressing the median nerve; in the second case that a cyst arising from the flexor tendon sheaths was compressing the median nerve; and in the third case that the median nerve was atrophic.

Section of the flexor retinaculum does not give rise to any functional impairment of the hand or wrist. This is probably because all powerful movements of the hand are made with the wrist in extension so that the flexor tendons cannot prolapse.[14, 17]

CASE REPORTS

Two cases are reported below:

Case 1

Mrs. de K., a 48-year-old housewife, related her trouble to a certain day 9 months before, when her maid stayed away from work and she had to do her own washing. That night she experienced an attack of numbness and discomfort in the right middle finger.

The condition progressed rapidly. The attacks became frequent and severe and the discomfort spread to involve the forefinger and later the thumb and wrist. Soon the attacks were very painful.

She found it difficult not only to localize the pain but also to describe it. She said the pain would stab from the wrist into the hand and fingers as far as the nails, which felt as though they were being pulled out. During the day there was numbness in the fingers and the severe pain came on only in nocturnal attacks. She could not stay in bed, and had to get up and walk about with the arm in the air. Pain was aggravated by hanging the arm down.

She noticed that the wringing of wet clothes would bring on discomfort and, if she used the hand a lot during the day, the pain would be worse that night.

No medicines or liniments made any difference to the pain.

She had been seen by an orthopaedic surgeon who, after taking X-rays of the cervical spine, diagnosed cervical disc disease. She was put in traction for 3 weeks without any relief.

Her left hand never gave her any trouble.

On examination it was found that there was atrophy of the lateral part of the thenar eminence and slight blunting of sensation over the palmar aspect of the thumb, forefinger and middle finger. With the transient ischaemia test, pain similar to her spontaneous attacks came on within 60 seconds. No other abnormality was noted.

The flexor retinaculum was sectioned through a transverse skin-incision.

Relief was dramatic. The night before operation she had her last attack of pain. Since operation she has not even had any discomfort. Her hand is strong and does not trouble her in any way.

Discussion. In all respects this is a typical case of far-advanced compression of the median nerve in the carpal tunnel. For a long time she was the victim of the non-specific diagnosis of 'neuritis', and later of the fashionable diagnosis of cervical disc disease. It is important to remember that cervical disc disease may co-exist with compression of the median nerve in the carpal tunnel and have nothing to do with the patients' symptoms.

Case 2

Mrs. P. a 58-year-old housewife, gave a history of 1 month's duration. She experienced attacks of pain in the right wrist, palm, and middle, ring and little fingers. From the onset the attacks rapidly became more frequent and more severe. The attacks occasionally came on during the day, but were particularly severe and frequent at night. She became afraid to go to bed at night, for pain would often come on within 10 minutes of lying down. Often she had to get up and walk about when pain came on. On one occasion the pain was so severe that she had to call out a doctor at night who gave her an injection for the pain. Pain would usually be slightly relieved by hanging the hand down. When pain was at its worst she noticed a tender spot in the middle of the front of the wrist.

Her left hand never gave her any trouble.

On examination no sensory blunting or motor weakness or wasting could be detected.

With the transient ischaemia test, pain similar to her spontaneous attacks came on within 60 seconds.

The flexor retinaculum was sectioned through a transverse skin-incision. Relief was dramatic. Since the operation was performed she has never had any pain or discomfort, and she says that her hand is in all respects normal.

Discussion. The unusual features here were that pain involved the little finger, that she did not complain of paraesthesia and that the history was short.

The paroxysmal nocturnal attacks, the absence of any demonstrable cause for the patient's complaints, together with the characteristic reaction to the transient ischaemia test, were enough to diagnose compression of the median nerve in the carpal tunnel and advise operation. The dramatic relief after section of the flexor retinaculum constitutes to us proof that the diagnosis was correct.

CONCLUSIONS

Compression of the median nerve in the carpal tunnel is characterized by paroxysmal nocturnal attacks of paraesthesia and pain which begin in one hand and may later involve the other hand. The symptoms are usually not accurately localized to the median-nerve distribution unless the patient is instructed to pay particular attention to the area involved during the attacks. In the early stages there is no demonstrable sensory or motor impairment. Later there is ill-defined sensory impairment in the median-nerve distribution in the fingers together with weakness and wasting of the muscles of the lateral part of the thenar eminence.

This condition, which is not uncommon, may be very

painful and incapacitating but relief is dramatic if the flexor retinaculum is sectioned.

SUMMARY

A short review is given of compression of the median nerve in the carpal tunnel or the so-called carpal-tunnel syndrome.

Two cases of the syndrome are reported and briefly discussed.

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